normal cells began to be more numerous than the pathological ones. The alterations he observed were enlargement of the Nissl bodies; peripheral and, later, complete chromatolysis; displacement of the nucleus; possibly diminution in the size of the nucleus; greater staining properties of the nucleus; and—when the degeneration was advanced—pale blue coloration of the protoplasm. He found also some degeneration of the posterior columns and posterior roots on the operated side, and in the region corresponding to the sciatic nerve. It may be stated, as a result of his experiments, that when the sciatic nerve in the rabbit is cut, many cells of the corresponding ganglia are altered, and most of these are later restored. The escape of certain cells, and the restoration of others, cannot be satisfactorily explained. While changes were found five days after the nerve was cut, degeneration of the posterior columns was not found until fifteen days after the operation; this would seem to indicate that the latter was the result of the former. The degeneration of the posterior columns was slight, and the spinal-ganglion cells which showed the highest degree of alteration were not numerous. Cassirer thinks the results of these experiments do not offer an explanation for the degeneration of tabes.

SPILLER.

70. EIN BEITRAG ZUR PATHOLOGISCHEN ANATOMIE DER EPILEPSIE (A Contribution to the Pathological Anatomy of Epilepsy). A. Alzheimer (Monatsschrift für Psychiatrie und Neurologie, vol. IV, 1898, p. 345).

Alzheimer describes two cases of epilepsy with dementia, in which he found degeneration of the medullated fibers of the cerebral cortex, especially of the tangential layer, proliferation of the glia, and destruction of ganglion cells. These changes were almost uniform throughout the cerebral cortex, but the vessels presented no important alteration. He believes from his investigations and those of others that cases of so-called genuine epilepsy exist, with characteristic pathological changes. In these cases there is macroscopically more or less sclerosis of the superficial cortical layer, and microscopically proliferation of the glia with a tendency to a normal arrangement, and considerable degeneration of nerve fibers and ganglion cells throughout the cortex. He believes that the nerve elements are primarily affected, and that the histological changes are sufficient to explain the dementia. Other cases of so-called genuine epilepsy exist without these characteristic changes, but the cases such as he describes, which he calls the Chaslin form of epilepsy, are not rare. It is probable that different diseases are included under what we now call epilepsy. The illustrations accompanying this paper are exceedingly interesting. SPILLER.

71. RECHERCHES SUR LES LÉSIONS DES CENTRES NERVEUX, CONSÉCUTIVES A L'HYPERTHERMIE EXPÉRIMENTALE ET A LA FIÈVRE (Studies of the Effects of Fever and Artificial Hyperthermia on the Nervous System). G. Marinesco (Revue neurologique, 7, 1899, p. 3).

Goldscheider and Flatau, Brasch, Moxter, Ewing and others have shown that, both in diseases attended with high temperatures, and in artificial raising of the temperature in animals, certain changes, similar in character, are present in the ganglion cells of the nervous system. In this present contribution, Marinesco comes to the conclusions that there are at least three stages in the development of complete chromatolysis, some of which he thinks have escaped other observers. In the first stage, the perinuclear elements and the nucleus show no marked variations; the prolongations, however, are not colored. These alterations are reparable. They are caused by high temperatures acting for a short time only. The second and third stages have been thoroughly reported upon by the observers above mentioned; but Marinesco thinks that these observers, for the most part, have not differentiated with sufficient care between lesions which result from high temperatures and lesions which might be produced by toxic agents in patients with high temperatures, as in pneumonia, typhoid and other infectious diseases. Thus in such cases, are the lesions found due solely to the increased temperature or to the toxic agent alone, or both? Marinesco believes that we are not yet in a position to decide definitely upon these questions. His general conclusions drawn from the present series of observations may be expressed as: (1) Temperatures below 40° C. (104° F.) in man, even if continued for several days, do not produce lesions as definite as in experimental hyperthermia in animals. (2) In febrile infectious cases, lesions may be found which do not belong to the temperature changes alone, since in experimental work in animals, similar changes do not seem to occur. (3) In cases in which the temperature has reached 41° C. (105.8° F.), and this has been maintained for several hours, lesions are found similar to those in experimental hyperthermia. JELLIFFE.

72. UNILATERAL CHANGES IN CEREBRAL HEMORRHAGE, EMBOLISM AND THROMBOSIS. Williamson (British Medical Journal, I, 1898, p. 1515).

In addition to the fundus changes currently conceded to be present with more or less rarity in sudden vascular lesions of the brain (albuminuric retinitis, embolism of central artery, narrowing of arteries, etc.), the author found retinal hemorrhages or dilatation of the retinal vessels on the side of the lesion in eight of thirteen cases of fatal cerebral hemorrhage or vascular occlusion, while the fundus on the opposite side was normal. In three cases both fundi were normal and in two albuminuric retinitis was present. PATRICK

CLINICAL NEUROLOGY.

73. ON BERI-BERI OCCURRING IN TEMPERATE CLIMATES. Conolly Norman (British Medical Journal, Vol. II, 1898).

After showing that the disease is not limited to the tropics and that in temperate climates it is particularly apt to occur in asylums, the author proceeds to describe the disease as observed by him at the Richmond Asylum, Dublin.

The first symptom observed was usually edema over the tibia, and generally cardiac disturbance was present from the very first. Dealing with a large number of cases, the diagnosis was roughly based on the following combination:

(1) Edema of the shins, without albuminuria; (2) tachycardia and cardiac irregularity; (3) pains in the legs; (4) anesthesia of the integuments of the legs. Of these it was somewhat difficult, owing to the mental condition of the patients, to estimate the degree of pain, etc., and often impossible to be sure of the existence of anesthesia.

Very frequently there was marked cardiac irritability and weakness and respiration was often thoracic, apparently from feeble action of the diaphragm.

Of the motor symptoms the earliest and most marked was paresis